



# MRI findings for unilateral sternoclavicular arthritis: differentiation between infectious arthritis and spondyloarthritis

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## Abstract

**Objectives** To analyze and identify magnetic resonance imaging (MRI) and clinical findings for the differentiation between infectious arthritis and spondyloarthritis in patients with unilateral sternoclavicular arthritis.

**Materials and methods** We retrospectively collected and evaluated the magnetic resonance (MR) images of 21 patients diagnosed with unilateral sternoclavicular arthritis, including 12 with infection and nine with spondyloarthritis, between 2004 and 2017. Capsular distension, extracapsular fluid collection, periarticular muscle edema, the prevalence and distribution of bone marrow edema, and the prevalence and size of bone erosions were assessed on the MR images. Clinical data were also reviewed.

**Results** Capsular distension was more prominent in patients with infectious arthritis than those with spondyloarthritis ( $p = 0.002$ ); extracapsular fluid collection and periarticular muscle edema were also more common in infectious arthritis than spondyloarthritis ( $p < 0.001$ , respectively); moreover, bone erosions were larger in infectious arthritis than spondyloarthritis ( $p = 0.023$ ). Other findings significantly associated with infectious arthritis included advanced age ( $p = 0.007$ ), an elevated C-reactive protein (CRP) level ( $p = 0.001$ ), and erythrocyte sedimentation rate (ESR) ( $p < 0.001$ ). The prevalence and distribution of bone marrow edema and the prevalence of bone erosions on MRI, the white blood cell count, and sex showed no significant differences between the two groups.

**Conclusions** Capsular distension, extracapsular fluid collection, periarticular muscle edema, and the size of bone erosions on MRI, as well as the age, CRP level, and ESR of patients, could be helpful for differentiating infectious arthritis from spondyloarthritis involving the sternoclavicular joint.

**Keywords** Arthritis, infectious · Magnetic resonance imaging · Spondyloarthritis · Sternoclavicular joint

## Introduction

The sternoclavicular joint is the diarthrodial articulation between the axial and appendicular skeletons. It is susceptible to the same disease processes that occur in joints, including degenerative arthritis, infection, and subluxation [1]. Moreover, it is a commonly involved site in patients with spondyloarthritis [2–5]. Although symmetrical involvement is seen in the majority of patients with spondyloarthritis, uni-

lateral involvement of the sternoclavicular joint has been noted in the early stages of ankylosing spondylitis, psoriatic arthritis, and reactive arthritis [2].

Differentiation between infectious arthritis and spondyloarthritis involving the sternoclavicular joint is very important for the selection of appropriate therapeutic approaches. Prompt diagnosis and treatment are crucial because untreated infection may lead to life-threatening consequences such as mediastinitis or empyema [1, 6, 7].

To our knowledge, no studies have attempted to differentiate between infectious arthritis and spondyloarthritis involving the sternoclavicular joint using magnetic resonance imaging (MRI). Accordingly, we designed the present study to analyze and identify MRI and clinical findings for the differentiation between infectious arthritis and spondyloarthritis in patients with unilateral sternoclavicular arthritis.

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## Materials and methods

This study was conducted according to the ethical standards of our medical institutional research committees as well as the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. Institutional review board approval was obtained before the initiation of this retrospective study, and the requirement for informed patient consent was waived.

Using the search term “sternoclavicular,” we retrospectively searched the radiology databases of our medical institutions for the period between January 1, 2004 and May 31, 2017. Our initial search yielded 178 patients. After a review of pathology and clinical reports, we excluded 157 patients with bilateral involvement, missing MRI data, a history of surgery or trauma, and underlying diseases such as rheumatoid arthritis and SAPHO (synovitis, acne, pustulosis, hyperostosis, and osteitis) syndrome. The final study group comprised 12 patients with infectious arthritis and nine with spondyloarthritis, both of which involved the unilateral sternoclavicular joint. Joint infection was confirmed by culture of aspirated specimens or blood or clinical follow-up studies. Five of the nine patients with spondyloarthritis fulfilled the Assessment of SpondyloArthritis International Society (ASAS) classification criteria for axial spondyloarthritis, while the remaining four were clinically diagnosed with undifferentiated spondyloarthritis on the basis of clinical examinations and MRI findings [8, 9].

MRI of the sternoclavicular joint was performed in the supine position. Because of the retrospective nature of our study, images were obtained with a variety of MRI scanners, including a 3.0-T MRI unit (Achieva; Philips Healthcare, Amsterdam, The Netherlands), a 1.5-T MRI unit (Achieva; Philips Healthcare, Amsterdam, The Netherlands), and a 1.5-T MRI unit (Signa; GE Healthcare, Erlangen, Germany). The imaging protocol and parameters also varied among cases. The imaging sequences included axial and coronal T1- and T2-weighted fast spin echo (FSE) sequences, short tau inversion recovery (STIR) or fat-suppressed T2-weighted sequences, and sagittal T2-weighted sequences. Fat-suppressed T1-weighted FSE sequences were acquired in the axial or coronal planes after the administration of a contrast agent in all 12 patients with infectious arthritis and eight patients with spondyloarthritis. An intravenous bolus injection of gadoterate meglumine (0.1 mmol/kg body weight; Dotarem; Guerbet, Paris, France) was administered at a rate of 2 ml/s for the postcontrast examination.

All MR images were randomly mixed and reviewed by two musculoskeletal radiologists (B.S.K. and H.S.S.) with 15 and 3 years, respectively, of clinical experience; they were blinded to the patients' history, findings of other imaging studies, and the final diagnosis. The following features were evaluated on the MR images: capsular distension, extracapsular fluid

collection, periarticular muscle edema, the prevalence and distribution of bone marrow edema, and the prevalence and size of bone erosions. Capsular distension was defined as fluid collection in the sternoclavicular joint and was measured perpendicular to the bone cortex on T2-weighted or STIR images. It was classified into two grades: grade 1,  $\leq 5$  mm and grade 2,  $> 5$  mm. Extracapsular fluid collection was defined as localized fluid collection outside the joint capsule, with or without rim enhancement, and it was evaluated on STIR or fat-suppressed T2-weighted sequences or contrast-enhanced, fat-suppressed T1-weighted images. Periarticular muscle edema was defined as increased signal intensity on STIR or fat-suppressed T2-weighted sequences. Bone marrow edema was defined as hyperintensity within the sternal manubrium or clavicle on STIR or fat-suppressed T2-weighted sequences or contrast-enhanced, fat-suppressed T1-weighted sequences. The distribution of bone marrow edema was divided into three categories: sternal manubrium-dominant, clavicular-dominant, and even. Bone erosion was defined as discontinuity in the bony cortex, with low signal intensity on T1-weighted images. The size of bone erosions was calculated from the greatest dimension and classified into two grades: grade 1, 1–5 mm; and grade 2,  $> 5$  mm. The number of bone erosions was not counted.

The following clinical data were reviewed for all patients: the C-reactive protein (CRP) level (normal, 0.0–1.0 mg/dl), erythrocyte sedimentation rate (ESR; normal, 0–15 mm/h), and white blood cell (WBC) count ( $>11,000$  cells/ $\mu$ l). We only analyzed clinical data obtained within 1 week of MRI examinations.

Statistical analyses were performed using SPSS version 20.0 (version 20.0, Inc., Chicago, IL, USA). The radiological and clinical data were reviewed and statistically compared between the two groups using Fisher's exact or Mann–Whitney *U* tests. A *p* value of  $< 0.05$  was considered statistically significant.

## Results

The final study group comprised six women and 15 men with an average age of 47.2 years (range, 16–80 years). In the infectious arthritis group, 58% (7/12) patients were men; the average age was 53.4 years (range, 36–65 years), and the average duration of symptom was 14.8 days (range, 1–60 days). In the spondyloarthritis group, 89% (8/9) patients were men; the average age was 39.0 years (range, 23–54 years), and the average duration of symptom was 83.2 days (range, 3–365 days). All 12 patients with infectious arthritis underwent joint aspiration: ultrasonography (US)-guided ( $n = 5$ ), open surgery ( $n = 5$ ), or both ( $n = 2$ ). Among them, biopsy of the sternoclavicular joint was also performed in five patients. None of the nine patients with spondyloarthritis

underwent joint aspiration or biopsy. The causative organism for infectious arthritis was identified by culture of tissue obtained from the sternoclavicular joint ( $n = 8$ ) and blood ( $n = 1$ ). The most common causative organism was *Staphylococcus aureus* ( $n = 4$ ), followed by *Streptococcus* species. ( $n = 2$ ), *Escherichia coli* ( $n = 2$ ), and *Mycobacterium tuberculosis* ( $n = 1$ ). The causative organism could not be identified for three patients, and the diagnosis of infectious arthritis was based on the clinical course of the disease and the response to antibiotic treatment.

The MRI findings for the two groups are summarized in Table 1. The two groups showed a significant difference with regard to capsular distension ( $p = 0.002$ ); grade 1 distension ( $\leq 5$  mm) was more common in the spondyloarthritis group than in the infectious arthritis group (8/9; 89%), whereas grade 2 distension ( $> 5$  mm) was more common in the latter group (10/12; 83%) than in the former group (Fig. 1a and b). The average amount of joint distension was 8.3 mm (range, 5.0–11.9 mm) in the infectious arthritis group and 3.1 mm (range, 1.5–5.5 mm) in the spondyloarthritis group.

Extracapsular fluid collection was observed in 92% (11/12) patients with infectious arthritis and none of the patients with spondyloarthritis, with a significant difference between groups ( $p < 0.001$ ) (Fig. 2a and b).

Periarticular muscle edema was observed significantly more frequently in the infectious arthritis group (12/12; 100%) than in the spondyloarthritis group (2/9; 22%), with the edema involving the pectoralis major or sternocleidomastoid muscle ( $p < 0.001$ ) (Fig. 2a). In addition, 50% (6/12) patients with infectious arthritis showed an abscess involving the pectoralis major or sternocleidomastoid muscle.

Bone marrow edema was noted in the sternal manubrium, clavicle, or both in 100% (12/12) patients with infectious arthritis and 67% (6/9) patients with spondyloarthritis, with no significant difference between groups (Fig. 3a and b). Even distribution was more common in the infectious arthritis group (9/12; 75%), while the clavicular-dominant distribution pattern was more common in the spondyloarthritis group (4/6; 67%; Fig. 4a and b). However, the two groups showed no

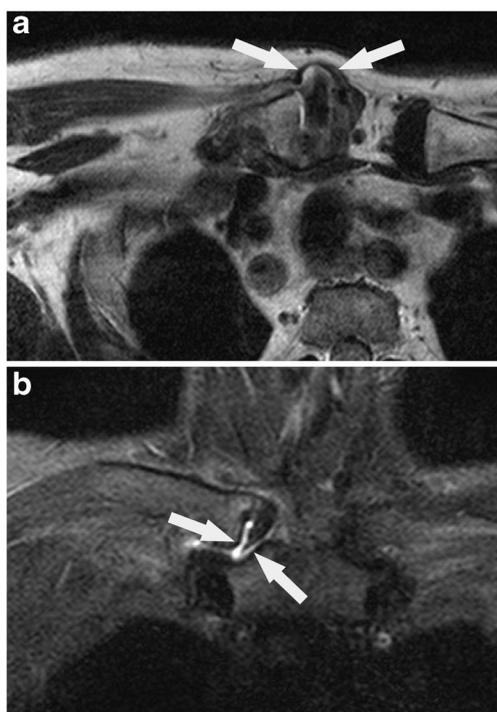
**Table 1** MRI findings of unilateral sternoclavicular arthritis in patients with infection and spondyloarthritis

| Finding  | Infection<br>( $n = 12$ ) | Spondyloarthritis<br>( $n = 9$ ) | $p$ value |
|--|---------------------------|----------------------------------|-----------|
| Capsular distension                            |                           |                                  | 0.002     |
| Grade 1 ( $\leq 5$ mm)                         | 2 (17)                    | 8 (89)                           |           |
| Grade 2 ( $> 5$ mm)                            | 10 (83)                   | 1 (11)                           |           |
| Extracapsular fluid collection                 |                           |                                  | $< 0.001$ |
| Absence  | 1 (8)                     | 9 (100)                          |           |
| Presence                                       | 11 (92)                   | 0 (0)                            |           |
| Periarticular muscle edema                     |                           |                                  | $< 0.001$ |
| Absence  | 0 (0)                     | 7 (78)                           |           |
| Presence                                       | 12 (100)                  | 2 (22)                           |           |
| Bone marrow edema                              |                           |                                  | 0.063     |
| Absence  | 0 (0)                     | 3 (33)                           |           |
| Presence                                       | 12 (100)                  | 6 (67)                           |           |
| Distribution of bone marrow edema <sup>a</sup> |                           |                                  | 0.141     |
| Clavicular dominance                           | 3 (25)                    | 4 (67)                           |           |
| Even distribution                              | 9 (75)                    | 2 (33)                           |           |
| Sternal manubrium dominance                    | 0 (0)                     | 0 (0)                            |           |
| Bone erosion                                   |                           |                                  | 0.063     |
| Absence  | 0 (0)                     | 3 (33)                           |           |
| Presence                                       | 12 (100)                  | 6 (67)                           |           |
| Size of bone erosion <sup>b</sup>              |                           |                                  | 0.023     |
| Grade 1 (1–5 mm)                               | 8 (67)                    | 5 (83)                           |           |
| Grade 2 ( $> 5$ mm)                            | 4 (33)                    | 1 (17)                           |           |

Data are number (%) of patients

<sup>a</sup> Distribution of bone marrow edema was assessed in six cases of spondyloarthritis with exclusion of three cases that were not noted bone marrow edema

<sup>b</sup> Size of bone erosion was assessed in six cases of spondyloarthritis with exclusion of three cases that were not noted bone erosion

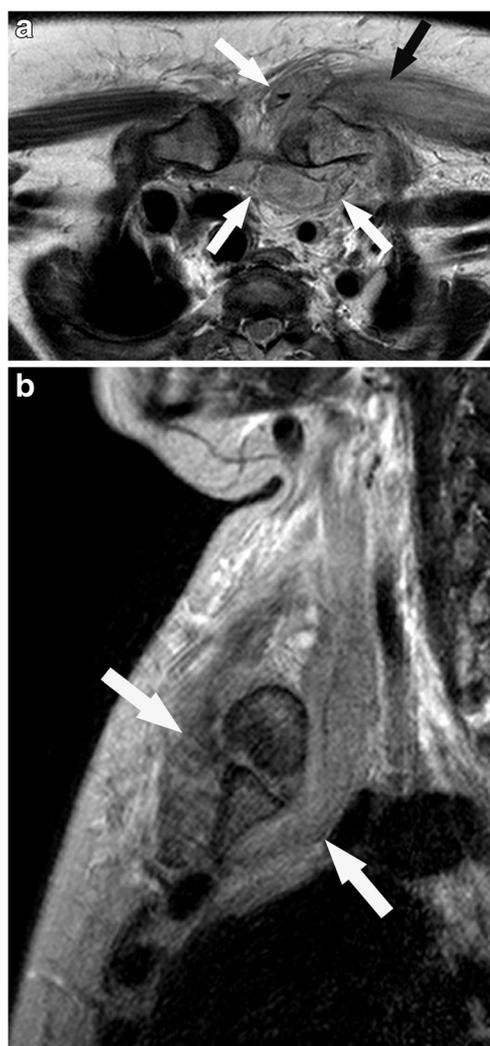


**Fig. 1** A 61-year-old woman with infectious arthritis involving the sternoclavicular joint (pathogen was not proven). **a** and **b** Axial T2-weighted (TR/TE, 2688/100) (**a**) and coronal STIR (TR/TE, 8491/70) (**b**) images show capsular distension with fluid accumulation in the right sternoclavicular joint (white arrows). TR: repetition time, TE: echo time, STIR: short tau inversion recovery

significant difference with regard to the distribution of bone marrow edema.

The prevalence of bone erosions was not significantly different between the infectious arthritis and spondyloarthritis groups. However, the size of bone erosions was significantly different ( $p = 0.023$ ) (Figs. 3a, b, and 5). The average size was 5.0 mm (range, 2.0–10.0 mm) in the infectious arthritis group and 2.3 mm (range, 1.0–5.6 mm) in the spondyloarthritis group.

The clinical data for the two groups are summarized in Table 2. Patients with infectious arthritis were significantly older than those with spondyloarthritis ( $p = 0.007$ ). There was no significant difference between groups with regard to sex. The serum CRP level was significantly higher in patients with infectious arthritis (mean, 11.8 mg/dl; range, 1.2–28.9 mg/dl) than in those with spondyloarthritis (mean, 1.3 mg/dl; range, 0.3–3.6 mg/dl) ( $p = 0.001$ ). The serum ESR was also significantly higher in patients with infectious arthritis (mean, 66.2 mm/h; range, 13–120 mm/h) than in those with spondyloarthritis (mean, 23 mm/h; range, 11–40 mm/h) ( $p < 0.001$ ). Although the WBC count was higher in the infectious arthritis group (mean, 12,248 cells/ $\mu$ l; range, 4330–20,250 cells/ $\mu$ l) than in the spondyloarthritis group (mean, 8344 cells/ $\mu$ l; range, 5300–13,100 cells/ $\mu$ l), the difference was not statistically significant.

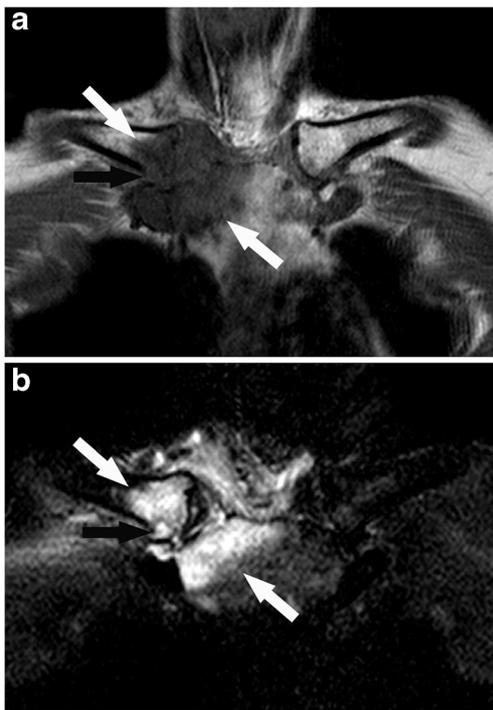


**Fig. 2** A 41-year-old woman with infectious arthritis involving the sternoclavicular joint, with *Staphylococcus aureus* identified as the causative pathogen. **a**, **b** Axial T2-weighted (TR/TE, 3000/80) (**a**) and sagittal T2-weighted (TR/TE, 3128/100) (**b**) images show extensive extracapsular fluid collection at the anterior and posterior aspects of the left sternoclavicular joint (white arrows). Periarticular muscle edema is noted in the left pectoralis major muscle (black arrow). TR: repetition time, TE: echo time

## Discussion

Our study results showed that capsular distension of  $> 5$  mm, extracapsular fluid collection, periarticular muscle edema, and large bone erosions on MRI, as well as advanced age and an elevated CRP level and ESR, were indicators of infectious arthritis involving the sternoclavicular joint.

MRI findings for sternoclavicular joint infection have been described in the literature; these include joint capsule distension, bone marrow edema, a heterogeneously enhancing soft tissue mass extending into adjacent soft tissues, and bone erosions [10–13]. With regard to spondyloarthritis of this joint, described MRI findings include bone marrow edema, bone erosion, and intra-articular effusion [3, 4]. Thus, several

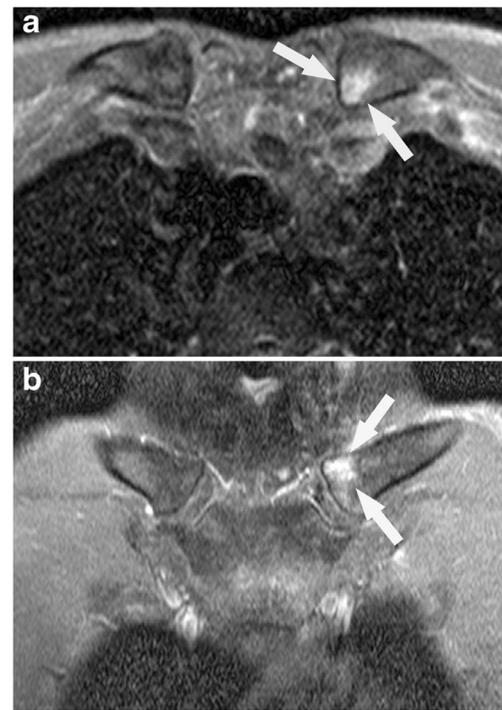


**Fig. 3** A 48-year-old woman with infectious arthritis involving the sternoclavicular joint, with *Staphylococcus aureus* identified as the causative pathogen. **a, b** Coronal T1-weighted (TR/TE, 482/10) (**a**) and STIR (TR/TE, 5248/80) (**b**) images show even distribution of bone marrow edema in the right sternoclavicular joint (*white arrows*). Bone erosion is noted in the medial portion of the right clavicle (*black arrows*). TR: repetition time, TE: echo time, STIR: short tau inversion recovery

MRI findings are shared by spondyloarthritis and infectious arthritis involving the sternoclavicular joint.

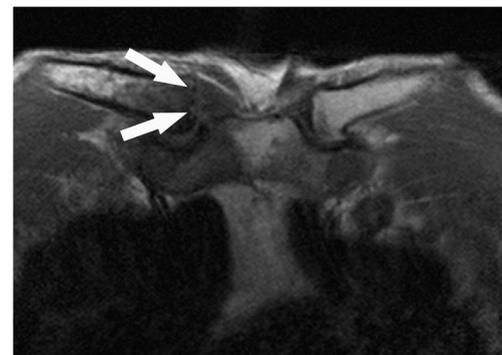
In the present study, capsular distension was more prominent in patients with infectious arthritis than in those with spondyloarthritis, while extracapsular fluid collection was observed only in patients with infectious arthritis. These findings may be related to the amount of joint fluid produced. Although extracapsular fluid collection, i.e., abscess, has been reported to occur in 20 to 80% cases in previous reports [6, 14], in the present study, 92% patients with infectious arthritis showed abscess formation in the anterior or posterior aspect of the sternoclavicular joint. According to Chen et al. [15], infection spread through superior capsule rupture would lead to erythema, swelling, and a neck abscess, whereas inferior capsule rupture would lead to retrosternal spread. On the other hand, inflammation associated with spondyloarthritis does not generally cross the anatomical borders, as observed in cases of sacroiliac joint involvement described by Navallas et al. [16]. These differences in joint capsule integrity between infectious arthritis and spondyloarthritis support the results of this study showing intra- and extracapsular fluid collections more frequently in infectious arthritis of the sternoclavicular joint.

Periarticular muscle edema was more commonly noted in the infectious arthritis group than in the spondyloarthritis group



**Fig. 4** A 36-year-old man with spondyloarthritis involving the left sternoclavicular joint. **a, b** Axial (**a**) and coronal (**b**) fat-suppressed, contrast-enhanced T1-weighted images (TR/TE, 500/9) of the left sternoclavicular joint show enhancement within the clavicle, which is consistent with bone marrow edema (*white arrows*). TR: repetition time, TE: echo time

in the present study. Edema extending to adjacent soft tissues, including muscles, has been reported in cases of infectious sternoclavicular arthritis [6, 12, 17, 18]. In our study, all patients with infectious sternoclavicular arthritis exhibited this finding. In addition, 50% cases showed an abscess in the pectoralis major or sternocleidomastoid muscle. Therefore, meticulous examination for periarticular muscle edema is required during evaluations of the sternoclavicular joint. However, two cases of spondyloarthritis also showed



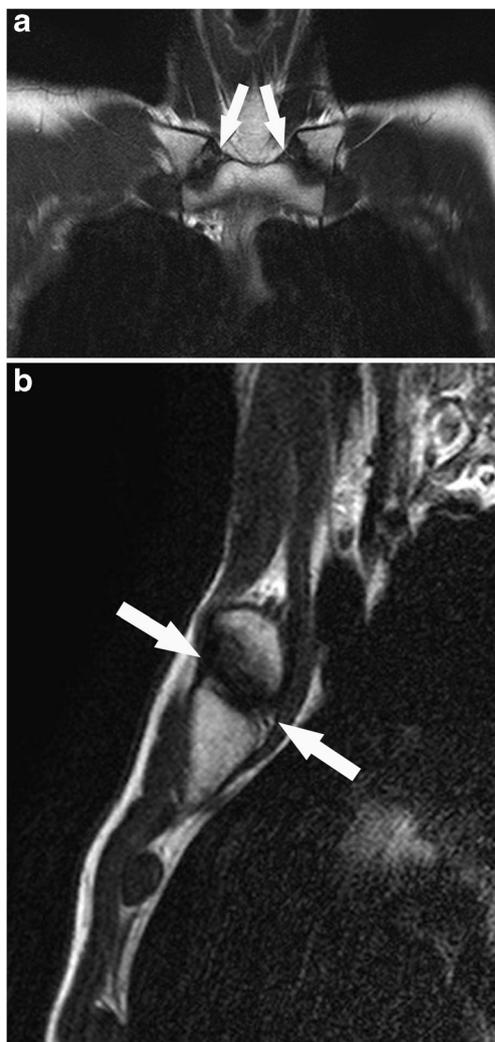
**Fig. 5** A 49-year-old man with spondyloarthritis involving the right sternoclavicular joint. A coronal T1-weighted image (TR/TE, 700/10) of the right sternoclavicular joint shows irregularity and small erosions in the medial portion of the clavicle (*white arrows*). TR: repetition time, TE: echo time

**Table 2** Clinical data of unilateral sternoclavicular arthritis in patients with infection and spondyloarthritis

| Finding              | Infection ( <i>n</i> = 12) | Spondyloarthritis ( <i>n</i> = 9) | <i>p</i> value |
|----------------------|----------------------------|-----------------------------------|----------------|
| Age (years)          | 53.4 ± 9.8                 | 39.0 ± 10.8                       | 0.007          |
| Sex (F:M)            | 5:7                        | 1:8                               | 0.178          |
| CRP (mg/dl) level    | 11.8 ± 9.4                 | 1.3 ± 1.2                         | 0.001          |
| ESR (mm/h)           | 66.2 ± 29.5                | 23.0 ± 10.1                       | <0.001         |
| WBC (cells/μl) count | 12,248 ± 5023              | 8344 ± 2504                       | 0.082          |

CRP C-reactive protein, ESR erythrocyte sedimentation rate, WBC white blood cells

periarticular muscle edema in our study. The fibrous capsule, i.e., the outer layer of the articular capsule that envelops the joint cavity, is firmly adhered to the periosteum of the articulating bones and is replaced by tendons or tendinous extensions from neighboring muscles at some sites [19] (Fig. 6a and b).



**Fig. 6** Normal sternoclavicular joints in a healthy 20-year-old man. **a** Coronal T2-weighted (TR/TE, 3004/100) image shows normal articular capsules of both sternoclavicular joints (*white arrows*). **b** Sagittal T2-weighted (TR/TE, 4159/100) image shows normal anterior and posterior articular capsules of both sternoclavicular joints (*white arrows*). TR: repetition time, TE: echo time

Inflammation of the fibrous capsule that envelops the sternoclavicular joint will extend to the periosteum of the sternum and clavicle; therefore, reactive edema of the adjacent muscles may occur as a secondary change.

In our study, bone erosions were larger in patients with infectious sternoclavicular arthritis than in those with spondyloarthritis. Bone erosion is seen not only after cartilaginous erosion but also after cartilage detachment due to insinuation of granulation tissue between cartilage and subchondral bone [20]. In a previous study using surface microscopy for the examination of macroscopically recognized articular surface defects, Rothschild et al. [21] mentioned that smaller “fronts of resorption” were commonly observed in inflammatory arthritis, including spondyloarthritis, while larger “zone of resorption” were commonly observed in tuberculosis and prominent “irregular resorption” was observed in pyogenic infection. These findings support the results of our study, which demonstrated that the size of bone erosions is significantly different between infectious arthritis and spondyloarthritis. Although evaluation of the erosions may have been influenced by curved joint surfaces and the slice thickness used, the size of bone erosions could be a helpful parameter for the differentiation of infectious arthritis from spondyloarthritis.

In the present study, the prevalence of bone marrow edema was similar in the infectious arthritis and spondyloarthritis groups. Although the difference was not significant, clavicular-dominant bone marrow edema was relatively more common in the spondyloarthritis group. In the sternoclavicular joint, unlike most synovial joints, the articular surfaces of the clavicle and manubrium are covered by fibrocartilage, which thicker over the clavicle than over the manubrium, instead of hyaline cartilage [22, 23]. Generally, spondyloarthritis does not affect the subchondral bone in the immediate vicinity of the articular cartilage that lines synovial joints. However, when such joints are lined with fibrocartilage instead of hyaline cartilage, the changes can be pronounced [24]. Also, Warth et al. [25] reported that approximately two-thirds of the medial clavicle was actually covered by articular cartilage, and portions of the medial clavicle that were not covered by articular cartilage served as attachment sites for the anterior and posterior capsular ligaments and intra-articular disc. These findings may explain the occurrence of bone marrow

edema in patients with spondyloarthritis involving the sternoclavicular joint. However, further studies with a large series of cases are needed on this matter.

In this study, the CRP level and ESR were significantly higher in the infectious arthritis group than in the spondyloarthritis group, whereas the WBC count showed no significant changes. Although clinical data have been shown to exhibit varied sensitivity and specificity for the diagnosis of infectious arthritis, most patients with this form of arthritis display elevated CRP levels and ESR, whereas peripheral blood WBC counts are often within normal limits in adults [7, 26–28]. In contrast to other systemic inflammatory diseases such as rheumatoid arthritis, spondyloarthritis may be associated with normal CRP levels and ESR in the majority of patients [29]. Therefore, the CRP level and ESR, not the WBC count, could be useful markers for differentiating between infectious arthritis and spondyloarthritis.

We observed that patients with infectious arthritis were significantly older than those with spondyloarthritis. Spondyloarthritis usually developed between the second and the fourth decades of life [30]. In contrast, the mean age for the development of infectious sternoclavicular arthritis is reported as 45 to 57 years (range, 11–88 years) [6, 7]. Therefore, considering that sternoclavicular arthritis develops in older patients, the likelihood of infectious arthritis is higher than that of spondyloarthritis in these patients.

In this study, there was no significant difference with regard to sex between the infectious arthritis and spondyloarthritis groups. Although men are more commonly affected by spondyloarthritis (especially ankylosing spondylitis) than women [31], the incidence of infectious sternoclavicular arthritis in men has also been reported to be as high as 70%–73% [6, 7].

In this study, aspiration of the sternoclavicular joint was performed in all 12 patients with infectious arthritis. On the contrary, the nine patients with spondyloarthritis did not undergo aspiration because spondyloarthritis was diagnosed using clinical diagnostic criteria. Further studies involving joint aspiration in patients with spondyloarthritis are required to reduce a clinical selection bias.

*S. aureus* was found to be the most common pathogen causing infectious arthritis in the present study. This finding was similar to that in previous studies [7, 32]. Sternoclavicular joint infection may occur via hematogenous spread from a distant source or contiguous spread from a nearby source of infection [12]. Sternoclavicular joint infection mainly affects immunocompromised individuals, such as those with existing infection, malignancy, rheumatic disease, diabetes, and alcohol and intravenous drug abusers, although it can also occur in previously healthy individuals [6, 33]. Among the 12 patients with infectious arthritis in the present study, four had diabetes mellitus and two had infection at other sites. The remaining six patients had no predisposing risk factors.

This study had several limitations. First was the retrospective design, and some medical records may have had missing or incorrect data. Second was the small number of patients in each group. Third, three different MRI scanners and protocols were used, which could have influenced the imaging findings. Fourth, a selection bias is likely to have resulted, since aspiration of the sternoclavicular joint was not performed in the nine patients with spondyloarthritis. Finally, when sagittal MR images are assessed, inadvertent visualization of signal intensity changes in facet joints or vertebral bodies in the thoracic spine may influence the analysis of findings.

In conclusion, the findings of the study suggest that capsular distension, extracapsular fluid collection, periarticular muscle edema, and the size of bone erosions on MRI, as well as the age, CRP level, and ESR of patients, could be helpful for differentiating infectious arthritis from spondyloarthritis involving the sternoclavicular joint.

## Compliance with ethical standards

**Conflict of interest** The authors declare that they have no conflicts of interest.

## References

- Higginbotham TO, Kuhn JE. Atraumatic disorders of the sternoclavicular joint. *J Am Acad Orthop Surg*. 2005;13:138–45.
- Guglielmi G, Scalzo G, Cascavilla A, Salaffi F, Grassi W. Imaging of the seronegative anterior chest wall (ACW) syndromes. *Clin Rheumatol*. 2008;27:815–21.
- Guglielmi G, Cascavilla A, Scalzo G, Salaffi F, Grassi W. Imaging of sternocostoclavicular joint in spondyloarthropathies and other rheumatic conditions. *Clin Exp Rheumatol*. 2009;27:402–8.
- Jurik AG, Zejden A, Lambert RG, et al. Pitfalls in MR morphology of the sterno-costo-clavicular region using whole-body MRI. *Clin Radiol*. 2013;68:785–91.
- Taccari E, Spadaro A, Ricciari V, Guerrisi R, Guerrisi V, Zoppini A. Sternoclavicular joint disease in psoriatic arthritis. *Ann Rheum Dis*. 1992;51:372–4.
- Bodker T, Tottrup M, Petersen KK, Jurik AG. Diagnostics of septic arthritis in the sternoclavicular region: 10 consecutive patients and literature review. *Acta Radiol*. 2013;54:67–74.
- Ross JJ, Shamsuddin H. Sternoclavicular septic arthritis: review of 180 cases. *Medicine (Baltimore)*. 2004;83:139–48.
- Rudwaleit M, van der Heijde D, Landewé R, et al. The development of Assessment of SpondyloArthritis International Society classification criteria for axial spondyloarthritis (part II): validation and final selection. *Ann Rheum Dis*. 2009;68:777–83.
- Zochling J, Brandt J, Braun J. The current concept of spondyloarthritis with special emphasis on undifferentiated spondyloarthritis. *Rheumatology*. 2005;44:1483–91.
- Johnson MC, Jacobson JA, Fessell DP, Kim SM, Brandon C, Caoili E. The sternoclavicular joint: can imaging differentiate infection from degenerative change? *Skelet Radiol*. 2010;39:551–8.
- Robinson CM, Jenkins PJ, Markham PE, Beggs I. Disorders of the sternoclavicular joint. *J Bone Joint Surg (Br)*. 2008;90:685–96.
- Harden SP, Argent JD, Blaquiere RM. Painful sclerosis of the medial end of the clavicle. *Clin Radiol*. 2004;59:992–9.

13. Klein MA, Spreitzer AM, Miro PA, Carrera GF. MR imaging of the abnormal sternoclavicular joint- pictorial essay. *Clin Imaging*. 1997;21:138–43.
14. Wohlgethan JR, Newberg AH, Reed JI. The risk of abscess from sternoclavicular septic arthritis. *J Rheumatol*. 1988;15:1302–6.
15. Chen WS, Wan YL, Lui CC, Lee TY, Wang KC. Extrapleural abscess secondary to infection of the sternoclavicular joint. Report of two cases. *J Bone Joint Surg Am*. 1993;75:1835–9.
16. Navallas M, Ares J, Beltrán B, Lisbona MP, Maymó J, Solano A. Sacroiliitis associated with axial spondyloarthritis: new concepts and latest trends. *Radiographics*. 2013;33:933–56.
17. Crisostomo RA, Laskowski ER, Bond JR, Agerter DC. Septic sternoclavicular joint: a case report. *Arch Phys Med Rehabil*. 2008;89:884–6.
18. Corey SA, Agger WA, Saterbak AT. Acromioclavicular septic arthritis and sternoclavicular septic arthritis with contiguous pyomyositis. *Clin Orthop Surg*. 2015;7:131–4.
19. Resnick D. Articular anatomy and histology. In: Resnick D, editor. *Diagnosis of bone and joint imaging*. 3rd ed. Philadelphia: Elsevier Saunders; 2002. p. 688–707.
20. Resnick D. Osteomyelitis, septic arthritis, and soft tissue infection: organisms. In: Resnick D, editor. *Diagnosis of bone and joint disorders*. 3rd ed. Philadelphia: Elsevier Saunders; 2002. p. 2510–624.
21. Rothschild BM. Differential diagnostic perspectives provided by en face microscopic examination of articular surface defects [published online ahead of print February 4, 2018]. *Clin Rheumatol*. <https://doi.org/10.1007/s10067-018-4001-x>.
22. Klein MA, Miro PA, Spreitzer AM, Carrera GF. MR imaging of the normal sternoclavicular joint: spectrum of findings. *AJR Am J Roentgenol*. 1995;165:391–3.
23. Brossmann J, Stäbler A, Preidler KW, Trudell D, Resnick D. Sternoclavicular joint: MR imaging-anatomic correlation. *Radiology*. 1996;198:193–8.
24. Benjamin M, McGonagle D. The anatomical basis for disease localisation in seronegative spondyloarthritis at entheses and related sites. *J Anat*. 2001;199:503–26.
25. Warth RJ, Millett P. Anatomy and biomechanics of the sternoclavicular joint. *Oper Tech Sports Med*. 2014;9:248–52.
26. Shirtliff ME, Mader JT. Acute septic arthritis. *Clin Microbiol Rev*. 2002;15:527–44.
27. Gupta MN, Sturrock RD, Field M. A prospective 2-year study of 75 patients with adult-onset septic arthritis. *Rheumatology*. 2001;40:24–30.
28. Li SF, Cassidy C, Chang C, Gharib S, Torres J. Diagnostic utility of laboratory tests in septic arthritis. *Emerg Med J*. 2007;24:75–7.
29. Rudwaleit M, Haibel H, Baraliakos X, et al. The early disease stage in axial spondylarthritis: results from the German Spondylarthritis inception cohort. *Arthritis Rheum*. 2009;60:717–27.
30. Braunwald E, Fauci AS, Kasper DL, Hauser SL, Longo DL, Jameson JL. Disorders of the immune system, connective tissue, and joints. In: Braunwald E, Fauci AS, Kasper DL, Hauser SL, Longo DL, Jameson JL, editors. *Harrison's principles of internal medicine*, 15th ed, part 12. New York: McGraw-Hill; 2002. p. 1805–2018.
31. Paparo F, Revelli M, Semprini A, et al. Seronegative spondyloarthropathies: what radiologists should know. *Radiol Med*. 2014;119:156–63.
32. Guillén Astete C, Aranda García Y, de la Casa Resino C, et al. Sternoclavicular septic arthritis: a series of 5 cases and review of the literature. *Reumatol Clin*. 2015;11:48–51.
33. Tanaka Y, Kato H, Shirai K, et al. Sternoclavicular joint septic arthritis with chest wall abscess in a healthy adult: a case report. *J Med Case Rep*. 2016;10:69.